model was then created to determine if the operative approach independently influenced outcome.

Results: There were 485 patients (385 CS [79%]; 100 AFB/MEVP [21%]). The cohorts iffered in that the AFB/MEVP group was younger (65.8 ± 12.5 vs 70.9 ± 9.7 years; $P < .001$), had more extent I/II aneurysms (66% vs 0.1%; $P = .0005$), and had more chronic dissections (30.3% vs 18.9%; $P = .0018$). Operative variables differed in that the AFB/MEVP cohort had longer operative times (434 ± 112 vs 324 ± 98 minutes; $P < .001$) and higher blood turnover (6028 ± 3473 vs 3581 ± 3111 mL; $P < .0001$). There was no difference in AFB/MEPB vs CS for the rate of intraoperative death (1.0% vs 0.05%; $P = .050$), length of intensive care unit stay (9.6 ± 8.6 vs 9.5 ± 12.3 days CS; $P = .095$), and hospital length of stay (19.9 ± 12.6 vs 21.6 ± 23.5 days; $P = .049$). The composite perioperative death and paraplegia rate was lower in the AFB/MEVP cohort (7% vs 19%; $P = .0004$). The multivariate model for predictors of the composite outcome showed that AFB/MEVP was protective (odds ratio, 0.039 [0.17, 0.9]; $P = .0028$). Long-term (4-year) survival was also improved in the AFB/MEVP group (73% ± 6% vs 60% ± 3%; $P = .0004$).

Conclusions: AFB/MEVP is an independent predictor of reduced perioperative death and paraplegia as well as improved long-term survival in patients undergoing repair of type I-III TAA and is the preferred operative strategy over CS.

Midterm Outcomes of Thoracic Endovascular Stent Repair for Chronic Type B Aortic Dissection With Aneurysmal Degeneration

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Introduction and objectives: The U.S. Food and Drug Administration has approved devices for endovascular management of thoracic aortic degenerative pathology (TEVAR); however, few data exist describing the outcome of TEVAR for aneurysms due to chronic type B aortic dissection (An-cTBAD). This study was undertaken to define the results of endovascular treatment of An-cTBAD.

Methods: A retrospective analysis of all patients treated for An-cTBAD was performed. Patient demographics, procedural outcomes, and anatomic covariates were aggregated. Reintervention and mortality were estimated using life-tables.

Results: Eighty patients (69 men [87%]) underwent TEVAR to treat An-cTBAD with mean ± standard deviation age 58 ± 13 years and median follow-up of 28 months (range 1-74 months). Median time from diagnosis of TBAD to TEVAR was 27 months (range, 1-74 months). Prior aortic root/arch replacement had occurred in 29% (n = 23) at a median interval of 28.5 months (range, 0.5-312 months). Mean aneurysm diameter was 62.0 ± 9.9 mm. Coverage occurred proximal to zone 3 in 75% (n = 60) of patients, and 24% (n = 19) underwent a carotid-subclavian bypass or arch debranching procedure. Spinal drains were used in 81% (preoperatively in 73% [n = 59]; and postoperatively in 8% [n = 6]). Length of stay was 6.5 ± 4.7 days, with a composite morbidity of 26% and in-hospital mortality of 2.5% (n = 2). Spinal cord ischemia developed in 9% (n = 7), with a permanent deficit observed in 6.2% (n = 5). Aneurysm diameter reduced or stabilized in 65%. The false lumen thrombosed completely within the thoracic aorta in 52%, and reintervention within the treated aortic segment was required in 16% (n = 13). At 1 year, freedom from reintervention was 75% and mortality was 83%.

Conclusions: TEVAR for An-cTBAD can be performed safely, but spinal cord ischemia rates may be higher than previously reported. Reintervention rates are acceptable; however, lack of uniform aneurysm diameter reduction and false lumen thrombosis underscore the need for longer-term follow-up and greater patient numbers to determine procedural durability and efficacy.

Blunt Thoracic Aortic Injury: A 14-Year Experience at a Level 1 Trauma Center

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Introduction and objectives: To review the natural history of blunt thoracic aortic trauma (BTAT) over a 14-year period at our level 1 trauma center and to compare open vs endovascular treatment.

Methods: All patients with BTAT presenting to a level 1 trauma center from 1998 to 2011 were included in a retrospective analysis. Multiple data points and short-term and moderate-term outcomes were ascertained by record review.

Results: The review identified 129 patients with BTAT. Of these, 32 (25%) were dead on arrival, 38 (29%) underwent an emergency department thoracotomy and died, 33 (25.5%) underwent open repair, 14 (11%) underwent endovascular repair, nine (7%) underwent a combination of procedures, and three (2%) were managed nonoperatively. Demographics, including injury severity score (average, 36) and revised trauma score (average, 9), were similar between the open and endovascular groups. The location of injury was also similar between groups (89% of injuries occurring at or ≤2 cm of the left subclavian artery). Rates of stroke, myocardial infarction, renal failure, and paralysis, length of stay (LOS), and 30-day mortality were decreased in the endovascular repair group compared with the open repair group (Table). Intraoperative time ($P = .053$), need for intraoperative red blood cell transfusion ($P < .001$), and estimated blood loss ($P < .001$) were all decreased in the endovascular group vs the open group. The average LOS for patients treated with endovascular repair was 15 vs 24 days in those treated with open repair ($P = .003$).

Conclusions: The incidence of BTAT is low but associated mortality is significant. During the 14-year period, there was a clear change in management preference from open repair to endovascular repair. Outcomes including stroke, myocardial infarction, renal failure, paralysis, LOS, and death seem to be reduced in the endovascular group.

Table. Comparison of complications between the open and endovascular repair groups

	Repair group, No. (%)		
Complications ≤30 days	*Open*	*Endovascular*	P
Death	14 (42)	1 (7)	.012
Myocardial infarction	2 (6)	0	.257
Stroke	3 (9)	1 (7)	.257
Renal failure requiring HD	3 (9)	0	.143
Paralysis	2 (6)	0	.473
Stent collapse	. . .	1 (7)	. . .

Thrombospondin-1 Differentially Regulates MicroRNAs in Vascular Smooth Muscle Cells

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Introduction and objectives: Thrombospondin-1 (TSP-1) is an important regulator of vascular smooth muscle cell (VSMC) physiology and gene expression. MicroRNAs (miRNA), which have emerged as potent regulators of cell function, are small molecules that regulate protein translation. miRNAs are be involved in intimal hyperplasia and atherosclerosis and are upregulated in the vasculature in diabetes. The purpose of this study was to identify miRNAs regulated by TSP-1 in VSMCs.

Methods: Human VSMCs were used. Both treatment groups consisted of three separate experiments, and the miRNA microarray was performed in triplicate for each sample. Cells were treated for 6 hours with basal media or TSP-1, both supplemented with 0.2% fetal bovine serum. Cells were snap frozen and RNA was extracted. An Affymetrix GeneChip miRNA array analysis was performed. Data was analyzed by analysis of variance with significance set at $P < .05$.

Results: TSP-1 upregulated 11 miRNAs and downregulated 23 miRNAs in VSMCs ($P < .05$). The most upregulated miRNA was miR-224 (1.57-fold). Also increased were miR-224 and miR-200c. The miRNA most downregulated by TSP-1 was miR-17*, which was decreased by 2.25-fold. Interestingly, 14 of the miRNAs altered were of the miRNA* strand.

Conclusions: The present study examined the effect of TSP-1 on miRNA expression in VSMCs. miRNAs regulate protein expression at the level of translation and may represent a significant mechanism by which TSP-1 regulates VSMC function. In addition, several of the miRNAs identified, such as miR-224 and miR-200c, have a role in vascular function. The miR-17 family, which exhibited reduced expression in this study, is known to inhibit cell proliferation and migration. We further found that several miRNA* strands were altered. The miRNA* strand, once thought to be the inactive complimentary strand of the mature miRNA, is now appreciated to regulate cellular function and may represent new targets for modifying TSP-1 signaling.

PARP Inhibition Modulates GAPDH Activity in a Diabetic Mouse Model of Hind Limb Ischemia–Reperfusion

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Introduction and objectives: Previous work has demonstrated that poly (ADP-ribose) polymerase (PARP) inhibition confers protection on skeletal muscle ischemia–reperfusion (IR) injury in diabetic mice. Published reports, including a model of renal IR injury in normal nondiabetic mice, have shown that poly (ADP-ribosylation) of the critical glycolytic enzyme